Hypereosinophilic syndrome in a cat

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Feline hypereosinophilic syndrome is a condition that occurs infrequently and is characterized by a persistent mature peripheral eosinophilia and eosinophilic infiltration of multiple organs. The condition responds poorly to chemotherapeutic agents.

This report details a case of hypereosinophilic syndrome in a cat with an unusual presentation. This cat had a healthy appetite throughout the course of its disease; all previous cases have been reported as having anorexia as a clinical finding.

In October 1995, a 6-year-old, spayed female, domestic shorthair was presented with a 4-month history of progressive weight loss, despite having a good appetite. Intermittent vomiting and diarrhea had begun 2 wk previously. The cat had not received any medication and had no travel history. Vaccinations were current.

On presentation, the cat was thin and weighed 3.5 kg. A firm mass was palpable in the cranial abdomen. There were no other remarkable physical findings.

A complete blood count and serum biochemical profile revealed a mature eosinophilia (5.664×10^9/L; reference range, 0.0 to 1.5×10^9/L) and an elevated amylase (1321 U/L; reference range, 490 to 1000 U/L) and creatine kinase (349 U/L; reference range 0 to 300 U/L). Mature eosinophils were present on a buffy coat analysis, but no mast cells were noted. Urine analysis was normal.

Tests for feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) (CITE Combo FeLV/FIV, IDEXX Laboratories, Westbrook, Maine, USA), heartworm antigen (Snap Canine Heartworm PF, IDEXX Laboratories), and fecal parasites were negative. Eosinophils were noted in the fecal smear. Resting thyroxine (T4) levels were within the normal range (31 nmol/L; reference range, 19 to 59 mmol/L).

Abdominal radiographs were unremarkable but an abdominal ultrasonagraph revealed a $5 \times 2 \times 3$ cm nodular, homogeneous, hyperechoic mass that incorporated large vessels. Cytological examination of an ultrasound-guided, fine needle aspirate of the mass revealed abundant eosinophils, low numbers of neutrophils, and a rare mast cell.

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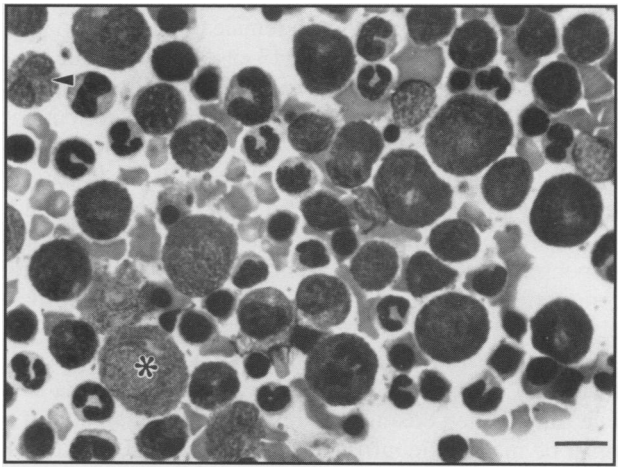

Figure 1. Bone marrow preparation showing hyperplasia due to increased eosinophilopoiesis. Note the orderly maturation and normal morphology of the eosinophils present. The starred cell is an example of a typical immature eosinophil. The arrowhead indicates a typical mature eosinophil. (Bar = 10 μm)

Bone marrow aspiration and cytological examination revealed expansion of the mitotic granulocyte pool due to orderly and enhanced eosinophilopoiesis. The granulocyte:erythroid ratio was 2:1 (Figure 1).

Exploratory laparotomy revealed an enlarged mesenteric lymph node, with other lymph nodes enlarged to a lesser degree. The pancreas was edematous and reddened. The intestinal walls appeared thickened. Other tissues were grossly normal. Significant histological findings included an eosinophilic lymphadenitis and diffuse eosinophilic jejunitis with fibrosis.

Based on these findings, we diagnosed hypereosinophilic syndrome (HES). The cat was discharged 1 wk postoperatively. Treatment consisted of systemic prednisone at a dose of 2 mg/kg body weight, PO, q12h. Complete blood counts were repeated on days 11 and 39 postpresentation; the peripheral eosinophil count remained elevated (6.78×10^9/L and 3.10×10^9/L, respectively). The eosinophil count in this cat was still in the lower range when compared with that of other cats with this syndrome, the majority of which have had eosinophil counts of 8×10^9/L or more. Vomiting and diarrhea worsened during a 3-week course of treatment, and the cat continued to lose weight despite having a ravenous appetite. Due to the poor prognosis and deteriorating condition, the owner elected to have the cat euthanized.

Gross postmortem examination showed enlargement of all mesenteric lymph nodes. Microscopically, there was eosinophilic infiltration of the mesenteric lymph nodes, submandibular lymph nodes, bone marrow, jejunum, spleen, and peripancreatic tissue.

Hypereosinophilic syndrome is a diagnosis of exclusion that is based on eosinophilia in the peripheral circulation and bone marrow, organ infiltration with eosinophils, and the absence of any recognizable cause for the eosinophilia (1–4). Hematological abnormalities other than persistent eosinophilia occur infrequently in HES (2,5).

Feline HES is a rare condition. To date, HES has not been reported in purebred cats (3). Ages of affected cats have ranged from 10 mo to 12 y with a mean age of 6 y (2,3). There appears to be a female predisposition (5). In one retrospective study, 9 of 13 cases were female (3).

The etiology of HES is unknown. The profound eosinophilia in this syndrome is believed to reflect an abnormal immunoregulatory ability or an alteration in the mechanism regulating eosinophil production. It is possible that an inappropriate response to an antigen could induce the eosinophilia by initiating an inflammatory response, which becomes systemic (6). Normal eosinophil production is dependent on T-lymphocytes and specific colony stimulating factors, IL-3 and IL-5 (1).

In the normal animal, only a small proportion of total body eosinophils are present in the circulation at any one time. When eosinophilia is recognized, an attempt must be made to identify causative factors. Conditions associated with the highest and most consistent eosinophilia include feline bronchial asthma, flea allergy dermatitis, eosinophilic granuloma complex, endoparasitism, feline eosinophilic enteritis, and mastocytoma (5,7). Other causes of eosinophilia include urinary tract disease, hyperthyroidism, immune-mediated disease, infectious diseases (panleukopenia, feline infectious peritonitis, and toxoplasma), and neoplastic disorders (lymphosarcoma) (1,3,6). In this case, these causes of eosinophilia were ruled out antemortem, or on postmortem examination.

Hypereosinophilic syndrome is characterized clinically by vomiting, diarrhea, anorexia, and weight loss (1,2,3,5–10). This undoubtedly reflects the high percentage of cases with gastrointestinal involvement (8). The cat in this report was not anorexic at any point during the course of the disease. It has been our experience that many cats with infiltrative bowel disease and malassimilation syndrome continue to eat.

The most frequently affected organs or tissues in cats with HES include bone marrow, spleen, lymph nodes, and the gastrointestinal tract. Less frequently involved tissues include skin, liver, lung, heart, pancreas, stomach, kidney, adrenal, and thyroid gland (2,3,5,8,9). The elevated amylase in this case may have been due to a chronic pancreatitis, as suggested by findings at the time of exploratory surgery and on histological examination of necropsy specimens.

Therapy for HES involves immunosuppressive doses of corticosteroids. Response to therapy is uniformly poor with survival times ranging from 1 wk to 3.5 y. The mean survival time from diagnosis to death is 7.5 wk (2,3,5,7,11). Cats succumb due to pathological changes in organs infiltrated with eosinophils.

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References

1. Center SA. Eosinophilia in the cat: A retrospective study of 312 cases (1975 to 1986). J Am Anim Hosp Assoc 1990; 26: 349–358.
2. Saxon B, Hendrick M, Waddle JR. Restrictive cardiomyopathy in a cat with hypereosinophilic syndrome. Can Vet J 1991; 32: 367–369.
3. Neer M. Hypereosinophilic syndrome in cats. Compend Contin Educ Small Anim Pract 1991; 13: 549–555.
4. Swenson CL, Carothers MA, Wellman ML, Kociba GJ. Eosinophilic leukemia in a cat with naturally acquired feline leukemia virus infection. J Am Anim Hosp Assoc 1993; 29: 497–501.
5. Huibregtse BA, Turner JL. Hypereosinophilic syndrome and eosinophilic leukemia: A comparison of 22 hypereosinophilic cats. J Am Anim Hosp Assoc 1994; 30: 591–599.
6. Bortnowski HB, Rosenthal RC. Gastrointestinal mast cell tumors and eosinophilia in two cats. J Am Anim Hosp Assoc 1992; 28: 271–275.
7. Plotnick A. Hypereosinophilic syndrome in a cat. Fel Pract 1994; 22: 28–31.
8. Hendrick M. A spectrum of hypereosinophilic syndromes exemplified by six cats with eosinophilic enteritis. Vet Pathol 1981; 18: 188–200.
9. Tobias SW. Hypereosinophilic syndrome in a cat. Mod Vet Pract 1985; 66: 1008–1009.
10. Herman GE, LaVonne MD. Eosinophilic enteritis in a specific pathogen-free cat. J Am Vet Med Assoc 1990; 197: 619–620.
11. Muir P, Gruffydd TJ, Brown PJ. Hypereosinophilic syndrome in a cat. Vet Rec 1993; 132: 358–359.